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Health Effects Related to Sewage Effluent Discharge into Fresh Water Environments

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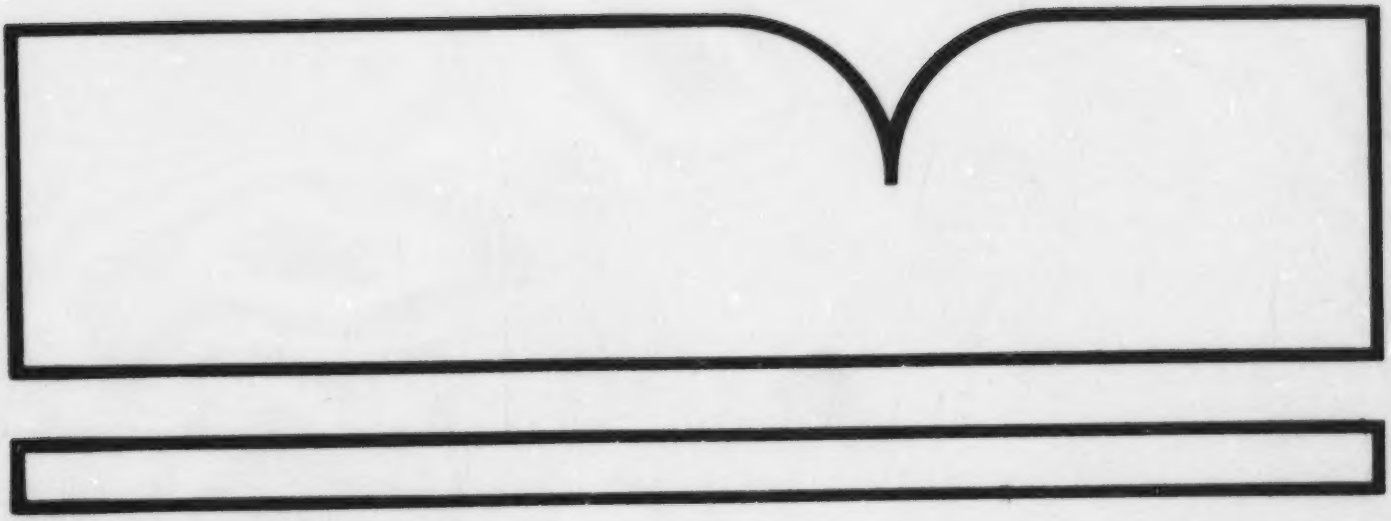

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HEALTH EFFECTS RELATED TO SEWAGE EFFLUENT DISCHARGE INTO FRESH WATER ENVIRONMENTS

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16. ABSTRACT

The relationship between sewage effluent particulates which reach bathing beaches and swimming-associated health effects was examined. A differential filtration procedure was used to quantify the density of particles greater than 3 microns in size carrying viable E. coli cells which served as a surrogate pathogen and the average number of E.coli per particle. Total particle density also was measured. Health effects were determined using a prospective epidemiological approach. The results of this two-year study show that there is an apparent relationship between swimming-associated illness rates and the density of microbial-bearing particulates at a freshwater beach. No relationship was observed between swimming-associated health effects and the total density of particles or the average number of viable bacterial units per particle. These results suggest that pathogens associated with illness in swimmers may circumvent disinfection and natural purification processes and reach bathing beaches by virture of their being encapsulated in fecal particulates.

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INTRODUCTION

The practice of disposing domestic wastewaters into streams, lakes and coastal waters carries with it an element of health risk for those individuals who use the receiving waters for leisure time activities, such as swimming, scuba diving and water skiing. One of the earliest recorded outbreaks of enteric illness due to swimming in sewage-contaminated water took place in Walmer, England, in 1908 (1). A group of young army recruits became ill with typhoid fever after a training exercise in an indoor seawater swimming pool which was usually filled on an incoming tide. Twenty of thirty-four cases were attributed to swimming in the pool. The source of the pathogen was sewage from two outfalls whose effluent was carried directly toward the pool intake with the rising tide. Eight of the first nine cases were members of a squad that had just completed their training in the swimming pool, a clear indication that it was a common source of the infections. Another well-documented outbreak of disease associated with swimming in sewage-contaminated water occurred a short distance downstream from Dubuque, Iowa, in 1975 (2). Thirty-one individuals were reported ill with shigellosis, and the only factor common to all the illnesses was swimming activity in the Mississippi River. Water samples from the river, examined some days after the peak of the outbreak, were found to contain high densities of coliforms. The pathogen which caused the illness, Shigella sonnei, was isolated from locations in the river where individuals who contracted shigellosis had been swimming.

A sewage treatment plant 17 miles upstream was suspected as the source of the pathogen, but this suspicion was never confirmed. Table 1 lists the two outbreaks just described and some other well-known outbreaks of infectious disease where there was an apparent linkage between sewage-contaminated water and swimming-associated illness. It should be pointed out that while the disease outbreak information in the table has frequently been used as proof of the connection between illness in swimmers and the accidental ingestion of contaminated bathing water, it should be accepted with a degree of skepticism, since much of the evidence is clearly circumstantial. This is especially true of the New York and New Haven outbreaks, where a sound relationship between swimming in polluted harbor water and typhoid fever was never clearly established (3,4). The etiological agent of illness in swimmers was isolated in only two of the outbreaks. Coxsacki A virus was isolated from the bathing water in the Niont, France, outbreak (5) and Salmonella paratyphi B was recovered from the river which served as a source of water for a riverside pool, and from swimmers who used the pool near Beccles, England (6). These two outbreaks present the clearest evidence that the accidental ingestion of sewage-polluted bathing water can result in gastrointestinal illness in swimmers.

Outbreaks of disease associated with swimming in sewage-contaminated water serve the very useful purpose of confirming what we intuitively believe to be true, i.e., if there are individuals in the community who have enteric disease, and their fecal wastes are discharged into aquatic environments without proper treatment, there is a probability that other individuals who use the receiving water for recreational purposes, such as swimming, will become ill if they accidentally ingest some of the water. Disease outbreak information also is useful for identifying new pathogen reservoirs

and in some cases, unrecognized diseases, but they provide little help for quantifying health hazards which result from the disposal of sewage into surface waters.

EPIDEMIOLOGICAL STUDIES

The use of epidemiological methods to establish possible relationships between swimming-associated illness and water quality was first attempted in the early 1950's. Stevenson (10) reported on a series of epidemiological studies which were conducted at marine and freshwater bathing beaches from 1948 to 1950. Significant swimming-associated health effects were not detected at marine bathing beaches, but an excess of gastrointestinal illnesses above what would be expected in the total study population was observed in swimmers at a freshwater beach on the Ohio River. Water samples taken during the course of the study were assayed for coliforms which at that time were the commonly accepted bacterial indicator for measuring the quality of the water. The geometric mean density of coliforms in the water samples was 2300 per 100 ml. Swimmers who used a public swimming pool containing high-quality water did not show an excess of gastrointestinal illness, as was observed in those who swam in a river. Stevenson concluded that there was a risk of enteric illness attributable to swimming in sewage-contaminated bathing water. This conclusion was tempered somewhat by the results reported by Moore in 1959 (11). He looked at the swimming activity of polio patients for the three weeks prior to the onset of the illness. Similar data was obtained from control subjects who had been matched to cases by age and sex. Moore did not find a higher frequency of swimming activity in the group that had contracted polio and he concluded that swimming in poor quality water was

not a risk factor for poliomyelitis. His conclusion frequently has been used to justify the case against extensive treatment and disinfection of sewage. A similar retrospective study conducted by D'Alessio and his colleagues in 1981 determined that there was an increased risk of enterovirus-caused illness in children who swam in lake water (12). Their results showed that children who contracted enteric illness swam significantly more often than those children who did not have enteric illness.

All of the epidemiological studies described above, though they were more elaborate than the disease outbreak investigations, were not very helpful for determining if a gradient existed between swimming-associated illness and decreasing water quality. In 1981, Cabelli, et al, (13) reported the results of a series of studies conducted at marine bathing beaches in New York City, Boston, and near New Orleans. The studies examined the differences in symptomatic illness rates between swimmers and nonswimmers on weekend days when the water quality was intensively monitored. The results of their studies clearly showed that as the quality of the bathing water decreased, the rate of swimming-associated gastrointestinal illness increased. The indicator they used to measure the quality of the water was a group of bacteria called enterococci, which are commonly found in the feces of warm-blooded animals. These indicator bacteria were used because they showed the strongest relationship to swimming-associated illness. The rate of swimming-associated gastroenteritis ranged from zero to 34.5 per thousand swimmers, and the mean enterococci densities ranged from 3.6 to 495 colony-forming units per 100 ml. Paradoxically, significant illness rates were sometimes associated with very high quality bathing waters.

FRESHWATER STUDIES

The detectable excess of gastrointestinal illnesses observed in the freshwater studies reported by Stevenson (10) and the results of the marine bathing beach studies showing a direct linear relationship between water quality and swimming-associated gastrointestinal illness described by Cabelli et al (13) indicated that further freshwater studies should yield significant results. In 1979, a series of freshwater trials were begun at two beaches on Lake Erie at Erie, Pennsylvania. The beaches are located on a long peninsula which extends into Lake Erie from west to east. (See Figure 1.) One beach is located on the northern perimeter of the peninsula (designated Beach A), and its waters are normally of good quality, except in situations when pollutants are carried along the shore by westerly winds. The second beach is located on the southeast shore of the eastern tip of the peninsula (designated Beach B). Beach B is approximately three-fourths of a mile northwest of the sewage outfall of the Erie sewage treatment plant. The sewage treatment plant serves a population of about 170,000 people and treats an average of about 45 million gallons of sewage per day, using an activated sludge process. The sewage is chlorinated before it is discharged into Lake Erie.

The design of the prospective cohort study described here was the same as that used by Cabelli et al (13) at marine bathing beaches. The salient features of the design are as follows:

1. Beach surveys and water quality monitoring were conducted only on weekends. This restriction was imposed because maximum beach usage occurred on those days. Whenever feasible, family groups were recruited for the study. This was done so that information on a number of individuals could be

obtained by questioning a group spokesperson, usually an adult member of the group, at the initial and follow-up interviews. Participants who swam in the five days before the trial weekend, or during the five days following the swimming experience were excluded from the study. Demographic data which included age, sex, ethnicity and socioeconomic status was obtained at the time of the beach interview.

2. Swimming was defined as completely immersing the head and body under water.
3. Eight to ten days following the initial interview the participants were contacted by telephone. After their eligibility was confirmed, they were questioned about the onset of any symptomatic illness that occurred after the swimming exposure. Information was obtained on gastrointestinal and respiratory symptomatology. Symptoms such as diarrhea, nausea, stomachache and vomiting were considered as gastrointestinal, and respiratory symptoms included sore throat, bad cough and chest cold. Fever was categorized under the heading "other" and it indicated a body temperature greater than 100 degrees F. Gastroenteritis was defined as having any one of the following unmistakable symptoms or combination of symptoms: (1) vomiting; (2) diarrhea with fever or a disabling condition such as remaining at home or in bed or seeking medical advice; and (3) stomachache or nausea accompanied by a fever.
4. A swimming-associated illness rate was determined by subtracting the symptomatic illness rate in nonswimmers from the symptomatic illness rate in swimmers.
5. The water quality was measured by sampling the water at two-hour intervals during peak swimming activity at three locations on each beach. The samples were assayed for enterococci using the method of Levin et al (14).

The results of the freshwater studies conducted on Lake Erie in 1979, 1980 and 1982 were very similar to those observed in the marine studies in three respects. First, swimmers appeared to have higher illness rates for all symptomatic illness compared to the rates in nonswimmers, although the rates were not statistically significant. This phenomenon also was observed in the marine recreational water studies (13) and in the freshwater studies reported by Stevenson (10). Second, there is a risk for swimming-associated gastroenteritis which is related to the quality of the water. This pollution-related effect is clearly shown in Table 2. At the relatively unpolluted Beach A, neither of the two summer trials yielded significant swimming-associated gastroenteritis symptom rates. However, at the more polluted Beach B, two of the three summer trials showed significant differences in the rate of gastroenteritis between swimmers and nonswimmers. The lack of corresponding significant swimming-associated rate differences for respiratory illness is indicative of the sewage-related nature of the illnesses observed in swimmers. These results clearly confirm the findings reported by Stevenson (10) in 1953, and they are very similar to the results obtained in the marine bathing beach studies described by Cabelli et al (13). Third, if there is indeed a relationship between gastroenteritis and water quality, then there should be a high correlation between the density of bacteria used to indicate the presence of fecal contamination and the degree of risk to swimmers. In the freshwater studies on Lake Erie, there was a high correlation between gastroenteritis and the density of enterococci in beach water samples. The strength of the relationship between enterococci and the relative risk for contracting gastroenteritis while swimming, or enterococci and the relative risk for respiratory illness was measured with the Spearman rank difference correlation coefficient (15). The correlation coefficient for the enterococci, gastroenteritis

variables was 0.90, which indicates that most of the increase in risk can be accounted for by the increase in indicator density (Table 2). The correlation coefficient for the enterococci, respiratory illness variables, on the other hand, was very small, 0.20, indicating a weak relationship between these two variables, a result that was not unexpected since it is unlikely that respiratory pathogens would be transmissible by ingestion even if they were present in domestic sewage. This strong relationship between enterococci, a bacterium intimately associated with the feces of humans and other warm-blooded animals, and gastroenteritis clearly establishes the health significance of discharging sewage into waters used for recreational activities.

The results of the studies conducted at Lake Erie beaches indicated that significant swimming-associated gastroenteritis occurred in reasonably good quality fresh water, a phenomenon which was also observed at marine beaches. This phenomenon occurred when the mean enterococci density was as low as 20 per 100 ml. The mean fecal coliform density in the same water samples was 60 per 100 ml. This fecal coliform density, which is about one-third of the standard recommended limit used by most states, is considered to be indicative of good water quality and yet it was associated with a significant swimming-associated difference in illness rates. In the marine bathing beach studies, significant swimming-associated gastroenteritis rates occurred when the enterococci density was as low as 11 per 100 ml. or the E. coli density was 15 per 100 ml. The significant gastroenteritis rates associated with swimming in good quality water are not easily rationalized. Viruses are thought to be the etiologic agent responsible for the swimming-associated gastroenteritis in swimmers (16), and this complicates the matter even more, since many viruses are not culturable and therefore their infectious doses are unknown. Although it was once assumed that one virus was sufficient to

initiate an infection (17,18), recent evidence indicates that multiple viral particles are required (19). Furthermore, it also is generally assumed that the maximum amount of water swallowed during swimming activity is about 10 ml. (20). These two factors imply that the causative agent of the gastroenteritis in swimmers is either highly virulent and therefore only a few viruses are required to initiate an illness, or the viruses occur in very high densities in the water and therefore only a small volume of water has to be ingested to receive an infective dose. The former premise is not amenable to experimental testing since the causative agent has not been identified. The second premise, however, can be subjected to testing if it is assumed that the high densities of pathogen are associated with or encapsulated in particles of fecal material. Such a study has been conducted at Lake Erie beaches.

PARTICLES AND GASTROENTERITIS IN SWIMMERS

The particulate study was carried out using the same health survey design previously described. The protocol for monitoring the microbiological quality of the water was modified so that bacterial cell-containing particles in a water sample could be quantified and the number of viable units per particle could be estimated. Since viral and bacterial pathogens are difficult to measure and their occurrence is sporadic, a bacterial surrogate was used to examine the potential relationship between swimming-associated illness and particulates in bathing water. Escherichia coli was selected as a pathogen surrogate for three reasons. First, this organism is a common inhabitant of the gastrointestinal tract and therefore it is intimately associated with fecal material, just as a pathogen would be. Second, E. coli,

as pointed out previously, is related to the incidence of gastrointestinal illness in swimmers, which implies that this organism is related to the presence of pathogens and may even physically occupy the same microenvironments as the pathogen. Enterococci, which also are closely related to the incidence of swimming-associated gastroenteritis, were not used as a surrogate because of their greater tendency to form chains and clumps. Third, a highly specific, facile enumeration technique was available for quantifying E. coli (21).

The procedure for obtaining estimates of the number of particles containing E. coli and the number of E. coli per particle is given in Figure 2. A portion of a water sample was assayed for E. coli with the standard technique used throughout the study and another portion was filtered through a 3 micron pore size polycarbonate membrane filter. A 3 micron pore size was used because it retained particles that were larger than a single E. coli cell. The assumption was made that all single, non-particle associated E. coli cells passed through the membrane into the filtrate. The filtrate was subsequently assayed using the standard technique. The colony count resulting from the original sample was called the standard estimate and that from the filtrate sample was called the filtrate estimate. The number of particles 3 microns or greater in size, containing at least one E. coli cell, was estimated by subtracting the filtrate estimate from the standard estimate.

The density of E. coli cells in the retentate was estimated by breaking up the particles and then assaying the resulting dispersed bacterial cells with the standard technique. This was accomplished by immersing the polycarbonate filter in a buffer solution containing a surfactant and then subjecting the filter containing solution to a brief sonication and blending treatment to desorb and disperse the E. coli cells. The average number of E. coli

per particle was estimated by dividing the total number of E. coli colonies obtained after the dispersion treatment by the number of E. coli associated particles.

Studies employing the particle enumeration procedures and the health effects survey methods were conducted during the summers of 1980 and 1982. The gastroenteritis illness rates observed in swimmers and nonswimmers in the summer of 1980 are given in Table 3. These data were used to calculate the relative risk in swimmers attributable to the water exposure. The relative risk was ordered from the smallest to the largest value and examined against the variables described above to determine if there was an association between increasing risk of illness and the measures of water quality. The values for the density of E. coli, the density of particles associated with E. coli colonies and the number of surrogate cells per particle, as well as the relative risk for each trial day are shown in Table 4. The Spearman rank difference correlation coefficient was used as a measure of the relative strength of association between the health effect parameter and each of the water quality variables (15). Table 5 indicates that there is a relationship between relative risk and the number of E. coli cells per particle, but that it is an inverse relationship, i.e., as the risk of gastroenteritis increases, the number of cells per particle decreases, an unlikely event if the surrogate were indeed a pathogen. The correlation between the risk of illness and the E. coli density, as obtained using normal monitoring procedures, is moderately low, indicating a positive but slight association. The correlation between the health effect risk and the density of surrogate colony associated particles, on the other hand, is a relatively strong one. The order of the relative strength of the three variables to swimming-associated risk was not unexpected. It is what might be anticipated if particle associated

pathogens were indeed responsible for swimming-associated gastroenteritis. Thus, the greater the density of particles containing multiple pathogens per volume of water, the greater would be the likelihood of a swimmer ingesting an infectious dose. This strong relationship between the density of E. coli associated particles and the risk of gastroenteritis would also explain the apparent lack of association between the number of surrogate bacteria per particle and the risk of illness. If the density of particles in the water is not great enough to insure a high probability of ingestion of at least a single particle by a swimmer, then it is irrelevant whether or not the particle contains a single pathogenic entity or a million.

The health effects and microbiological data collected during the summer of 1982 are shown in Tables 6 and 7. A new characteristic was substituted for the number of viable bacteria per particle in the second year of the study. The density of all particles less than 50 microns, those carrying viable bacteria as well as those not associated with microbial cells, was quantified using an electronic particle counter. This new parameter and two of the variables examined in 1980 were analyzed using the Spearman rank difference correlation coefficient described above. The results of this analysis are given in Table 8. Once again the density of E. coli associated particles showed the strongest relationship to the risk of gastrointestinal illness, as it did in the 1980 study. The relationship between E. coli density and swimming-associated risk of illness was quite strong as compared to the slight association observed in 1980. The total particle count showed a negative relationship to the illness risk very similar to the relationship observed between illness risk and E. coli associated particles in 1980. This finding also points to the fact that risk of gastrointestinal illness is indeed associated with particles of fecal material and not particulates in

general.

The rationale for the trials which examined the relationship between health effects in swimmers and particulates in the water was based on the hypothesis that an infectious dose of a pathogen could be ingested in a small volume of water if the pathogens were adsorbed to or encapsulated in fecal material. Thus, a single particle might contain hundreds or even thousands of bacterial or viral pathogens. This hypothesis was attractive because it could account for many of the known factors which were incompatible with some of the observations made from data collected in the epidemiological-microbiological studies at marine and freshwater bathing beaches. For instance, it was known that many virus particles were required for an infective dose, and that the amount of water ingested during swimming activity was small, and yet a significant number of illnesses were occurring in spite of the fact that the water was judged to be of high quality by current standards. The hypothesis is also supported by available evidence which indicates that bacteria and viruses adsorbed to or encapsulated in particles are protected from environmental stresses and the effects of disinfection. Hejkal et al (22) have shown that polioviruses encapsulated in fecal material are more resistant to chlorination than nonencapsulated viruses. Similarly, a number of investigators (23,24,25) have presented evidence that particle associated bacteria and viruses survive longer than non-particle associated bacteria and viruses. These two factors could account for the occurrence of gastrointestinal illness at beaches affected by sewage treatment plant effluents that not only have been disinfected, but which sometimes must travel considerable distances before reaching a beach.

Although the research work on this problem is not complete, the preliminary evidence presented here strongly supports the hypothesis that

swimming-associated gastrointestinal illness observed in individuals bathing in good quality water is at least in part the result of particles which contain an infective dose of a pathogen.

IMPLICATIONS OF PARTICULATE ASSOCIATED ILLNESS IN SWIMMERS

The apparent relationship between swimming-associated illness and the density of bacteria laden particulates in bathing waters implies that some form of intervention might be practiced which could influence the gastrointestinal illness rate in swimmers. For instance, decreasing the density of microorganism associated particles in the water should lead to a corresponding decrease in the relative risk for gastroenteritis in swimmers. The break-up of particles in wastewater effluents would have a two-fold effect on any potential pathogens that might be contained in the particles. First, the exposed bacteria and viruses would be much more susceptible to disinfection, and, second, the pathogens discharged into receiving waters would be fully exposed to physical and biological factors which promote biological decay in aquatic environments. The results of this study also imply that incomplete intervention could have an adverse effect on the rate of swimming-associated health effects. Although the data are limited, they clearly indicate that breaking up large particles that contain high numbers of viable units per particle to smaller particles which still contain an infective dose of pathogen may actually increase the rate of gastroenteritis in swimmers. However, this conclusion must remain speculative until confirmed in future studies.

CONCLUSIONS

The results of the Lake Erie bathing beach studies on the relationship between swimming-associated illness and water quality lead to the following conclusions.

The presence of sewage wastes in recreational waters, as measured with bacterial indicators, is a significant risk factor for gastrointestinal illness associated with swimming activity.

The rate of swimming-associated gastrointestinal illness is correlated with water quality; that is, as the water quality decreases, the rate of illness in swimmers increases.

Statistically significant swimming-associated illness rates occur in water considered to be high quality according to currently accepted standards. Preliminary evidence from studies examining the effect of particulates in water on the health of swimmers indicates that the swimming-associated illness in individuals bathing in good quality water may be the result of particulates laden with high numbers of pathogens. This finding, if confirmed in ongoing studies, could be useful in planning intervention measures that might appreciably lower the rate of gastrointestinal illness in swimmers.

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TABLE 1

OUTBREAKS OF DISEASE ASSOCIATED WITH SWIMMING ACTIVITY

Year	Reference	Type of Water	Disease or Agent	Water Quality
1908	1	Sea	Typhoid	Poor
1921	3	Sea	Typhoid	Poor
1932	4	Sea	Typhoid	Poor
1947	6	Fresh	Salmonellosis	Poor
1958	7	Sea	Typhoid	Poor
1973	8	Fresh	Coxsacki B Virus	Unknown
1974	5	Fresh	Coxsacki A Virus	Poor
1978	2	Fresh	Shigellosis	Poor
1979	9	Fresh	Viral Enteritis	Unknown

TABLE 2

RELATIVE RISK RATIOS AND MICROBIAL INDICATOR DENSITIES

FOR LAKE ERIE BEACHES, 1979, 1980 AND 1982

Year	Beach	Relative Risk[1]		Mean[2] Enterococci Density/100 ml
		Gastroenteritis	Respiratory Illness	
1979				
	A	1.2	1.2	5
	B	1.3	1.3	13
1980				
	A	1.4	0.68+	25
	B	2.3*	1.3	71
1982				
	B	1.8*	1.3	20

Correlation Coefficient[3]		0.90	0.20	

[1]Illness rate in swimmers divided by the illness rate in nonswimmers.

[2]Geometric mean.

[3]Spearman's rank difference correlation coefficient - A measure of the strength of relationship of relative risk to indicator density.

*Swimmer illness rate statistically greater than the nonswimmer illness rate ($P < 0.05$)

+Nonswimmer illness rate statistically greater than the swimmer illness rate ($P < 0.05$)

TABLE 3

GASTROENTERITIS ILLNESS RATES OF SWIMMERS AND NONSWIMMERS

BY TRIAL DAY AT BEACH B, 1980

Trial Day	Swimmers		Nonswimmers	
	Number of Participants	% Ill	Number of Participants	% Ill
1	345	4.3	139	4.3
2	300	3.0	109	1.8
3	268	3.4	92	1.1
4	255	1.6	235	0.4

TABLE 4

HEALTH EFFECTS RISK, WATER QUALITY LEVEL,

AND PARTICLE-RELATED MEASUREMENTS FOR BEACH B, 1980

Relative Risk	E. coli Per 100 ml	Colony Associated Particles Per 100 ml	E. coli Density/Particle
1.0	138	30	1500
1.6	286	154	158
3.1	109	64	426
3.7	624	484	17

TABLE 5

CORRELATION OF SWIMMER-ASSOCIATED GASTROENTERITIS
WITH WATER QUALITY INDICATOR DENSITY AND PARTICLE RELATED VARIABLES

Comparison	Correlation Coefficient [1]
Gastroenteritis vs No. *E. coli*/Particle	-0.80
Gastroenteritis vs *E. coli* Density/100 ml	0.40
Gastroenteritis vs Density of *E. coli* Associated Particles/100 ml	0.80

[1] Spearman's rank difference correlation coefficient

TABLE 6

GASTROENTERITIS ILLNESS RATES OF SWIMMERS AND NONSWIMMERS

BY TRIAL DAY AT BEACH B, 1982

Trial Day	Swimmers		Nonswimmers	
	Number of Participants	% Ill	Number of Participants	% Ill
1	335	1.8	147	1.4
2	422	2.1	203	1.5
3	352	2.3	137	1.5
4	451	6.9	141	4.3
5	460	3.3	174	1.1
6	495	4.0	128	0.7

TABLE 7

HEALTH EFFECTS RISK, WATER QUALITY LEVEL,

AND PARTICLE-RELATED MEASUREMENTS FOR BEACH B, 1982

Relative Risk	E. coli Per 100 ml	Colony Associated Particles Per 100 ml	Particle Density/100 ml
1.32	66	20	2030
1.44	60	32	1908
1.56	72	46	1521
1.62	65	24	1689
2.84	142	64	1435
5.17	135	104	1863

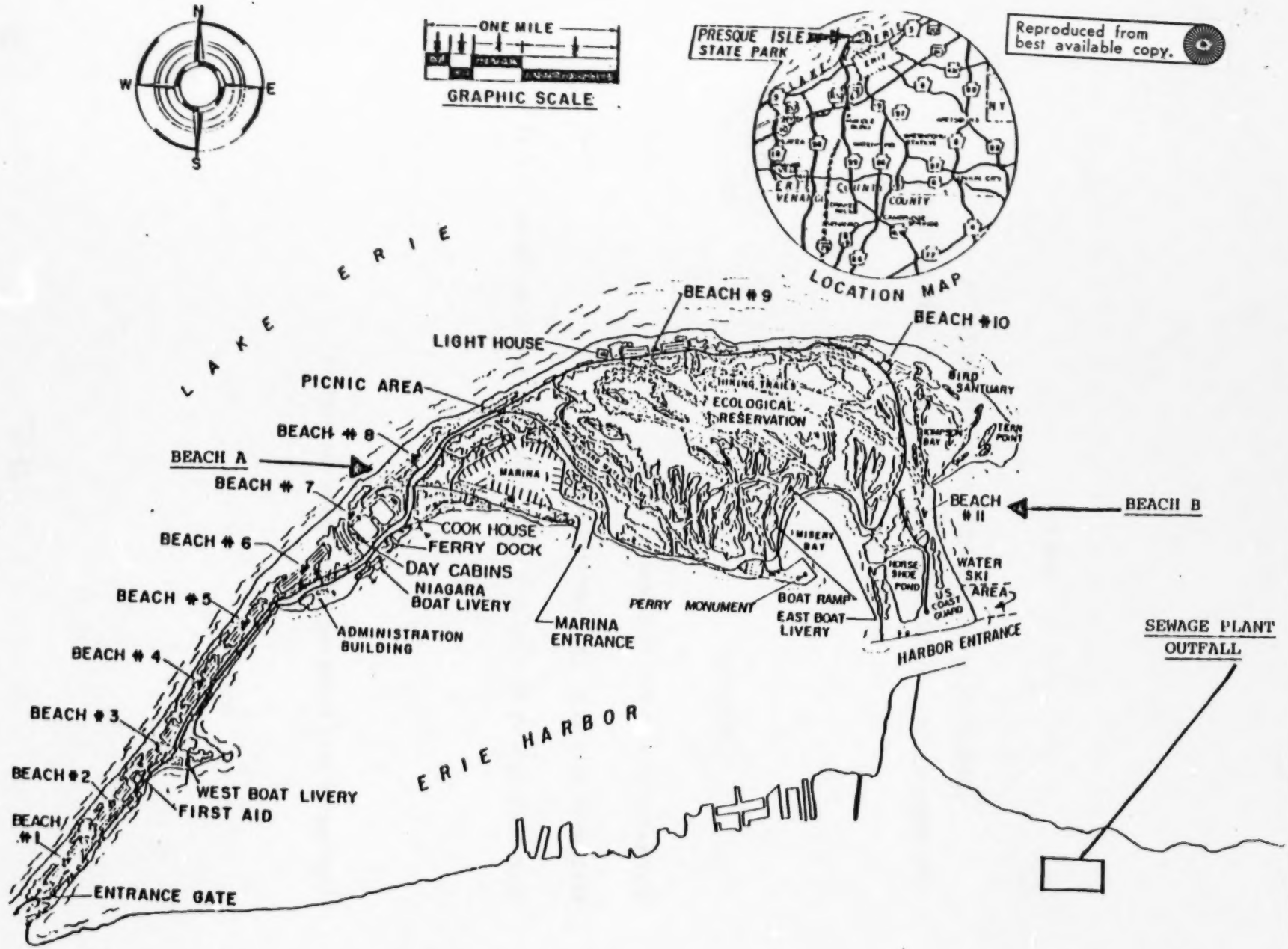
TABLE 8

CORRELATION OF SWIMMER-ASSOCIATED GASTROENTERITIS

WITH WATER QUALITY INDICATOR DENSITY AND PARTICLE RELATED VARIABLES

Comparison	Correlation Coefficient [1]
Gastroenteritis vs Particle Density/100 ml	-0.60
Gastroenteritis vs *E. coli* Density/100 ml	0.66
Gastroenteritis vs Density of *E. coli* Associated Particles/100 ml	0.71

[1] Spearman's rank difference correlation coefficient



PRESQUE ISLE PENINSULA

Figure 1.

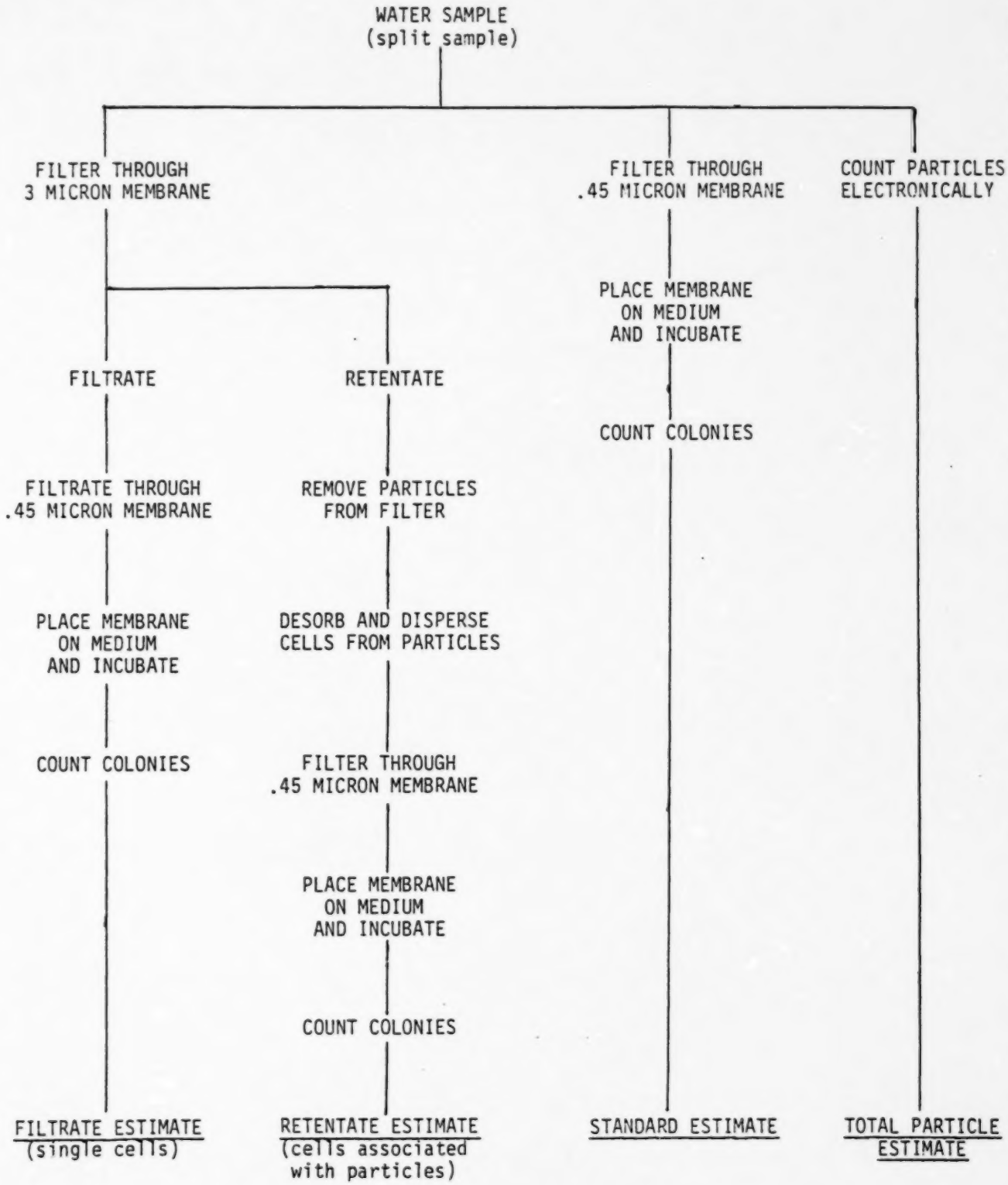


Figure 2. Sample Treatment Protocol